

Hæmodynamic Changes During Continuous Exercise

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Since many cardiac patients complain of symptoms only during exercise, it seems reasonable for an exercise test to be included in the assessment of their disability. The timing of measurements taken during the exercise period is obviously of importance and it is common practice to assume a "steady-state" after the initial three or four minutes. Readings obtained during this "steady-state" are then accepted as control values for comparison with later measurements made, for example, after the patient has been given 100 per cent oxygen to breathe for several minutes. Changes in the control values as exercise is prolonged have been studied only infrequently, and represent an obvious source of error in assessing the effects of oxygen or other drugs. In this study we report the results obtained when patients continued steady exercise for at least 30 minutes with no drugs being given.

SUBJECTS AND METHODS

Seven symptom-free patients with moderately loud ejection systolic murmurs in the absence of depressed sternum were investigated (Table I); two of these patients (Nos. 6 and 7) were known to have mild pulmonary stenosis and were having routine follow-up investigations. The eventual diagnosis in the other five patients was of a normal heart. Since all subjects, including those with pulmonary valve gradients, behaved in an identical fashion, the results have been considered together.

The exercise test was in all cases carried out as an out-patient procedure; a fine nylon catheter† was floated into the pulmonary artery from an antecubital vein, the position being checked by a pressure trace displayed on an oscilloscope. One millimetre outer diameter teflon tubing‡ was introduced into the brachial artery by the Seldinger technique and was advanced into the aorta.

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These "micro-catheters" enable investigations to be performed on out-patients with no need for x-ray screening. The procedures carry very little risk and the only complications in our laboratory over several years have been slight bruising around the arterial puncture and the occasional provocation of ectopic beats when the fine nylon catheter passes through the right ventricle.

Consolidated Electrodynamics strain gauges (type 4-326-L212) were used, and pressures and electrocardiograms were recorded on a Sanborn direct writer. Blood oxygen content was estimated with a Brinkman hæmoreflector calibrated against Van Slyke analyses. Oxygen uptake and carbon dioxide production was recorded every eight seconds at rest and during exercise by means of a Junkalor "Spirolyt" paramagnetic oxygen analyser which has been fully described previously (Barold, Burkart, and Sowton, 1966), and cardiac output was calculated by the Fick principle. Left ventricular work was calculated as the product of cardiac output and mean aortic ejection pressure. Tension time index (Sarnoff *et al.*, 1958) was calculated as the product of the mean aortic ejection pressure, the heart rate, and the ejection time measured from the aortic pressure trace. Total peripheral vascular resistance and total pulmonary vascular resistance were calculated from the cardiac output and the mean aortic and pulmonary artery pressures respectively.

TABLE I
DETAILS OF THE SEVEN SUBJECTS STUDIED

Subject	Sex	Age (yr.)	Height (cm.)	Weight (kg.)	Hb (g./100 ml.)	Erect, resting RV-PA gradient (mm. Hg)
1	M	21	176	68	16.5	0
2	M	52	173	77.5	15.0	0
3	M	13	149	43	14.5	0
4	M	25	183	68.5	16.3	7
5	M	24	177	73	16.2	3
6	F	16	160	60	13.8	8
7	F	20	165	63	14.0	40

Subjects 6 and 7 were known to have mild pulmonary valve stenosis. Patients 4 and 5 were judged to have normal hearts.

TABLE II

MEAN VALUES FOR THE WHOLE GROUP OF SUBJECTS WHEN SUPINE AT REST, ERECT AT REST, AND DURING PROLONGED EXERCISE ON A BICYCLE ERGOMETER AT 300 KPM./MIN.

	No. of subjects	Position		Time (min.) since onset of exercise									
		Supine	Erect	2	5	7-8	9-10	12	14-15	20	24-27	30	
Aortic mean pressure (mm. Hg)	5	93	103	116	115	111	109	107	106	107	106	103	
Pulmonary artery mean pressure (mm. Hg)	6	17.1	14.1	19.3	17.1	17.6	17.1	17.1	16.8	16.6	16.5	16.3	
Heart rate (beats/min.)	6	89	100	122	129	137	138	140	140	144	146	146	
Cardiac output (l./min.)	5	6.9	5.2	10.9	10.8	10.7	10.7	10.5	10.5	10.9	10.6	10.6	
Stroke volume	5	78	52	89	84	78	78	75	76	76	75	75	
LV work/min. (kg.m.)	5	10.3	8.8	19.9	20.1	19.2	19.2	17.8	18.7	18.7	17.2	17.2	
Tension time index mm. Hg (sec./min.)	5	2560	2890	3740	4010	3860	3860	3540	3670	3670	3420	3420	
AV-O ₂ -diff. (vol./100 ml.)	5	46.1	59.1	92.6	98.0	96.8	96.8	94.3	96.1	96.1	95.1	95.1	
Oxygen uptake (ml./min.)	5	319	307	992	1050	1037	1037	1024	1042	1042	1052	1052	
Total peripheral resistance units	5	13.6	20.7	10.8	10.7	10.4	10.4	10.4	9.9	9.9	9.6	9.6	
Total pulmonary resistance units	5	2.5	2.7	1.8	1.7	1.7	1.7	1.7	1.7	1.7	1.6	1.6	

Pressures and electrocardiograms were recorded at rest in both supine and erect positions and also every two to three minutes during exercise on a bicycle ergometer (Holmgren and Mattsson, 1954); after 15 minutes of exercise, measurements were made every 5 minutes for a total of 30 to 40 minutes. Blood samples were taken at rest in both positions and during exercise every 3 to 4 minutes for the first 15 minutes, and then at 20, 30, and 40 minutes. For all subjects the exercise level remained unchanged at 300 kpm./min. throughout the investigation. One patient (No. 2) with a normal heart was unable to continue exercise beyond 18 minutes because of pain in a leg due to an old injury; his results have not been included in the mean values but the responses of all parameters were qualitatively the same as for the other six patients. In one 13-year-old boy (No. 3) no arterial catheter was introduced and only pulmonary artery pressure and heart rate figures are available. The remaining five patients had an average age of 21 years (range 16-25). One of the two patients previously diagnosed as having pulmonary stenosis had right ventricular-pulmonary artery gradients of 40 mm. Hg at rest and on exercise, and the other had gradients of only 8 mm. Hg at rest and 5 mm. Hg on effort. Two of the patients judged to have normal hearts had small right ventricular to pulmonary artery gradients of up to 10 mm. Hg on exercise.

The zero level for pressure recordings in the supine position was the mid-chest, and the zero level in the upright position was the point of insertion of the fourth rib into the sternum.

RESULTS

The detailed results are given in Table II which presents the mean figures for the whole group.

Aortic Pressure. When the patients adopted the erect position the mean pressure increased by 10 mm. Hg; during exercise it rose abruptly to reach a peak level two minutes after the onset of exercise and then fell slowly to reach the pre-exercise level after 30 minutes (Fig.).

Pulmonary Artery Pressure. The pressure fell by 3 mm. Hg when the patients adopted the erect position. During exercise the peak level was reached within two minutes but the pressure then slowly fell to approach its original value after 30 minutes, though by this time it was still slightly raised above the pre-exercise level.

Heart Rate. The mean resting level was 89 beats a minute in the supine position and this high figure presumably reflects some apprehension on the part of the subjects. The mean level increased to 100 beats a minute when the patients were sitting at rest on the ergometer; during exercise the heart rate increased fairly rapidly to 137 beats a minute after 8 minutes but then continued to rise very slowly to a final level of 146 beats a minute at the end of the exercise period (Fig.).

Cardiac Output. The mean resting cardiac output in the supine position was 6.9 litres a minute and this fell to 5.2 litres a minute in the erect position. During exercise, the peak value of 10.9 litres a minute was reached within two minutes and the cardiac output stayed nearly constant with a slight tendency to fall towards the end of the exercise test.

Stroke Volume. The mean stroke volume in the supine position was 78 ml. and this fell to 52 ml. in the erect position. After two minutes of exercise a peak value of 89 ml. was reached, but after eight minutes the stroke volume had fallen to the same value as in the supine resting position; a further decrease continued during the next 25 minutes to a final level of 73 ml. (Fig.).

Left Ventricular Work. The left ventricular work fell by 14.5 per cent when the subjects assumed the erect position. It then increased considerably

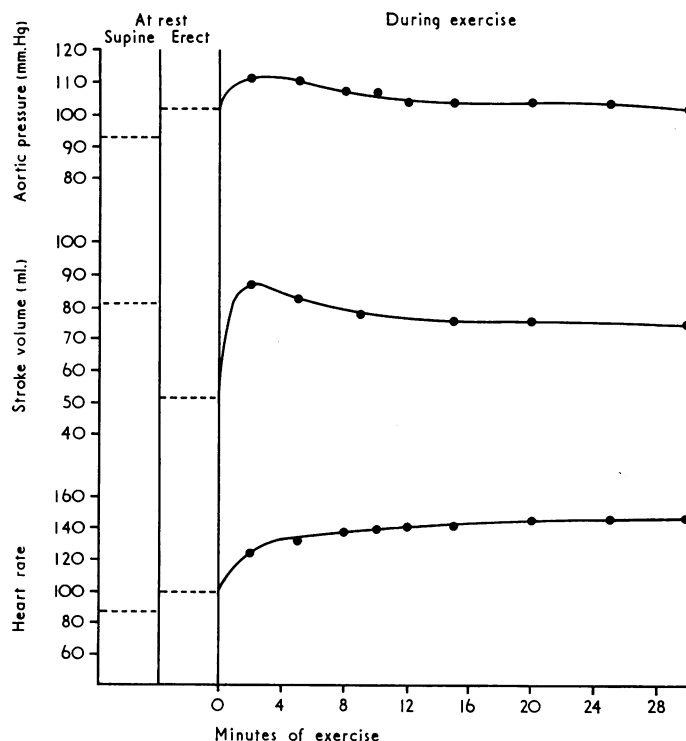


FIG.—Changes in mean aortic pressure, stroke volume, and heart rate for five subjects during continuous exercise at 300 kpm./minute on a bicycle ergometer. The zero level in the supine position is mid-chest. Zero level in the erect position is the insertion of the fourth rib to the sternum.

with exercise to reach a peak after about two minutes, with a slow decline following during the whole period of exercise. If values obtained during the erect position only are considered, the left ventricular work increased during exercise at 300 kpm./min. to 230 per cent of the resting value with reduction to 196 per cent of the resting value after 30 minutes of exercise.

Tension Time Index. This index increased by about 12 per cent when the patients assumed the erect position. It rapidly increased further at the onset of exercise, with the five-minute reading producing the highest levels; there was then a slow reduction throughout the whole period of exercise to a final level which was 19 per cent higher than the erect resting value.

Arteriovenous Oxygen Difference. This increased from a value of 46.1 ml./100 ml. to 59.1 ml./100 ml., with the change in posture. During exercise, there was a rapid increase which was maximum in the five-minute measurements; there was then a slow reduction throughout the remaining exercise period.

Oxygen Uptake. The oxygen uptake increased rapidly with exercise to reach a maximum between three and four minutes after the onset of exercise. The level then remained approximately constant for the remaining exercise period.

Total Peripheral Resistance. The resistance increased by about 50 per cent with the change to the upright position. It fell rapidly with exercise and had approximately halved within two minutes; it then continued to decrease slowly throughout the exercise period.

Total Pulmonary Resistance. This increased only slightly in the erect position but fell abruptly within the first two minutes of exercise. There was then a slow continued decrease, so that after 30 minutes of exercise the resistance averaged 60 per cent of the initial value.

DISCUSSION

The alteration in posture from lying supine to sitting erect on the bicycle ergometer was accompanied by a mean fall in cardiac output of 25 per

cent; the stroke volume fell by 36 per cent and there was a 12 per cent increase in the heart rate. These changes are in agreement with previous reports (Bevegård, Holmgren, and Jonsson, 1960) and emphasize the necessity for the body position during the control measurements to be accurately stated. In this study all percentage changes are referred to the resting supine control level unless stated otherwise.

The alterations in blood pressure with the change in posture are difficult to interpret because of uncertainty over the zero level. The aortic pressure apparently increased by 11 per cent and the pulmonary artery pressure fell by 18 per cent with the assumption of the upright position, so that errors in the zero level cannot entirely account for the postural changes in pressure. The alterations in cardiac output and stroke volume were relatively considerably greater than the changes in pressure so that it may be tentatively concluded that the left ventricular external work is reduced in the erect compared with the supine position; our figures suggest that the fall is approximately one-seventh of the supine resting value, and a similar fall occurred in the tension time index. It is unlikely that the tension time index bears the same relation to myocardial oxygen consumption when the subject is in the erect position as when in the supine, and so it does not necessarily follow that myocardial oxygen uptake is reduced in the erect posture though this seems likely.

The aortic pressure rose sharply with the onset of exercise to reach a maximum increase of 13 mm. Hg at about 2 minutes. After 8 minutes the increase was only 9 mm. Hg and after a further 4 minutes the increase was only 4 mm. Hg, showing that the timing of measurements from the onset of exercise may greatly influence the results and that the term "after having reached the steady rate" is insufficiently precise. Within 30 minutes of exercise the mean aortic pressures in our subjects had returned to the pre-exercise level despite the unchanged work load, a finding in keeping with the results reported by Holmgren (1956). The reason for this progressive fall has not been fully explained but it is probably due to increasing peripheral vasodilatation. The pulmonary artery pressure behaved similarly to the aortic during prolonged exercise, though the changes were less pronounced. The mean results for our group show that the pulmonary artery pressure had not returned to the pre-exercise level (erect position) but in the two patients who were exercised for longer periods the pressure continued to fall to reach the pre-exercise level. Similar results have been reported by Ekelund and Holmgren (1964).

The rapid increase of cardiac output to reach its

peak after only two minutes can be related to the low exercise level and the comparative fitness of this group of young people. After this initial rise cardiac output remained fairly constant throughout the entire duration of exercise, and this result is similar to that reported by Cobb and Johnson (1963) who exercised their subjects on a treadmill. Levy, Tabakin, and Hanson (1961) found the cardiac output in normal men during exercise was steady only for short periods and reported considerable variations in several subjects when exercise was prolonged, but Grimby, Nilsson, and Sanne (1963), using a dye-dilution technique with the subjects exercising on a bicycle ergometer, found a considerably smaller variation and obtained results similar to those of the present study.

The heart rate was relatively steady after a few minutes but then continued to rise slowly throughout the entire period of the exercise both in this and other studies. The stroke volume therefore fell slowly but continuously throughout the entire period of exercise, and this has been a constant finding in investigations employing higher work load on a bicycle ergometer (Ekelund and Holmgren, 1964; Grimby *et al.*, 1963) as well as treadmill exercise (Cobb and Johnson, 1963). The continual fall in both aortic and pulmonary artery pressures despite the maintained cardiac output indicates a progressive fall in both systemic and pulmonary total vascular resistances during exercise, and this finding applies to subjects exercised either in the supine (Sancetta and Rakita, 1957) or in the erect position (Ekelund and Holmgren, 1964).

The alterations during exercise in left ventricular external work per minute and in tension time index were not comparable. The relative increase in left ventricular work was considerably greater than that in tension time index (referred to erect resting value), though both indices were at their highest between two and five minutes of exercise. Despite the uncertainty concerning interpretation of changes in these indices between rest and exercise, it seems reasonable to conclude that both the work of the heart and the myocardial oxygen uptake decrease from this early peak when exercise at a constant work load is prolonged. These changes may account for the ability of some patients with ischaemic heart disease to "walk through" an attack of angina.

SUMMARY

In six symptom-free young subjects an alteration in posture from supine to erect resulted in a 25 per cent fall of cardiac output and a 33 per cent fall in stroke volume.

On exercise at 300 kpm./minute there was an initial rise in aortic and pulmonary pressure, but

these then fell progressively to approach the erect resting level after thirty minutes of continuous exercise. The heart rate increased slowly throughout the period of exercise, but the cardiac output and oxygen uptake remained almost unchanged once the steady state had been reached.

Cardiac work and tension time index decreased as exercise was prolonged.

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